Effect of chronic dissection on early and late outcomes after descending thoracic and thoracoabdominal aneurysm repair

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Objective: **Although chronic aortic dissection (CD) has traditionally been considered a predictor of perioperative morbidity and mortality after descending thoracic/thoracoabdominal aneurysm repair (thoracoabdominal aortic aneurysm [TAA]), recent reports have rejected this assertion. Still, few contemporary studies document late outcomes after TAA for CD, which is the goal of this study.**

Methods: **From August 1987 to December 2005, 480 patients underwent TAA; 73 (15%) CD and 407 (85%) degenerative aneurysms (DA). Operative management consisted of a clamp-and-sew technique with adjuncts in 53 (78%) CD and 355 (93%) DA patients ($P < .001$). Epidural cooling was used to prevent spinal cord injury (SCI) in 51 (70%) CD and 214 (53%) DA patients ($P = .007$). Study end points included perioperative SCI/mortality, freedom from reintervention, and long-term survival.**

Results: **CD patients were younger (mean age 64.5 years CD vs 72.5 years DA, $P < .001$) and more frequently had a family history of aneurysmal disease (23% CD vs 6% DA, $P < .001$). Forty-three (59%) CD patients had elective TAA (vs 322 (79%) DA, $P = .001$). Eleven (15%) CD patients had Marfan's syndrome (vs 0% DA, $P < .001$), and 17 (23%) CD patients had a prior arch or ascending aortic repair (vs 16 [4%] DA, $P < .001$). CD patients were more likely to have Crawford type I & II thoracoabdominal aneurysms (44 [60%] vs 120 [29%] DA, $P < .001$), while only two (3%) CD patients had type IV aneurysms (vs 99 [24%] DA). There was no difference in perioperative mortality between the two groups (11% CD vs 8.6% DA, $P = .52$), nor was there a difference in flaccid paralysis, which occurred in five (7%) CD and 22 (5%) DA patients ($P = .92$). At 5 years, 70% of CD patients were free from reintervention versus 74% of DA ($P = .36$). The actuarial survival was 53% and 32% at 5 and 10 years for CD versus 47% and 17% for DA ($P = .07$).**

Conclusions: **Despite increased operative complexity, CD does not appear to increase perioperative SCI or mortality after TAA when compared with DA. Long-term freedom from aneurysm-related reintervention is similar for both groups as is survival, despite patients with CD being of younger age at presentation. (J Vasc Surg 2011;53:600-7.)**

Patients with chronic type B aortic dissections (CD) are at risk of aneurysmal dilation of the outer wall of the false lumen, with an unpredictable temporal sequence.[1] It has been estimated that over 50% of patients with CD will rupture or require thoracoabdominal aortic aneurysm (TAA) resection within 4 years of their initial presentation.[2-4] These patients typically require a more complex operative repair than patients with degenerative aneurysms (DA), which can lead to longer cross-clamp durations and increased renal and spinal cord ischemia. Indeed, in Crawford's sentinel report of 605 patients, CD was associated with a significantly higher risk of paraplegia than patients with degenerative aneurysms (30.4% vs 7.4%, $P < .0001$),[5] and a subsequent review of the world's literature 10 years later also identified CD as a predictor of increased spinal cord ischemic (SCI) risk.[3] This in turn has often been related to maintenance of intercostal vessel patency in aneurysms of chronic dissection versus degenerative etiology.[6]

The addition of intra-procedural adjuncts such as distal aortic perfusion via atrial-femoral bypass,[7,8] epidural cooling (EC),[9] and the monitoring of motor evoked potentials[10] has improved the expected paraplegia rates after repair of types I–III TAA. In several recent series that have applied these adjuncts to TAA repair, dissection was no longer identified as a predictor of adverse outcomes on multivariate analysis.[11,12] However, little has been documented regarding the late outcomes of TAA repair in patients with CD. The goal of this study is to compare the results of TAA repair between patients with CD and DA in a contemporary series with long-term follow-up.

METHODS

Clinical, demographic, and operative details of all patients undergoing TAA repair at our institution have been prospectively collected and maintained in a database

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throughout the study interval (August 1, 1987-December 30, 2005). A review of this database identified 480 patients who underwent open descending thoracic or thoracoabdominal aneurysm repair during the study period, which included those who underwent both elective and emergent repair. The distribution of procedures is as follows: 52 (11%) descending thoracic, 82 (17%) type I TAA, 82 (17%) type II TAA, 163 (34%) type III TAA, and 101 (21%) type IV TAA. Patients were stratified into two cohorts based on the underlying cause of the aneurysm (CD vs DA). The 73 (15%) patients who presented with chronic dissection comprise the study cohort. The Institutional Review Board of the Massachusetts General Hospital approved this protocol, and individual consent was waived.

Pre- and intraoperative variables as well as perioperative outcomes were prospectively compiled over the study period. Chronic obstructive pulmonary disease (COPD) was determined by preoperative pulmonary function tests that were obtained on most patients. Urgent operation was defined as either rupture or presentation with acute aortic syndrome necessitating invasive monitoring in an intensive care unit and operative repair within 48 hours of admission. Renal insufficiency was defined as a creatinine >1.8 mg/dl, and patients were considered to have a history of heart disease even if they had previously been revascularized with coronary artery angioplasty or bypass grafting.

Operative conduct

The clamp and sew technique with or without neuroprotective adjuncts was used in 90% of cases. Distal aortic perfusion was reserved for patients with anticipated technically complex proximal anastomosis or significant renal insufficiency and was used in approximately 10% of the procedures. EC has been used for spinal cord protection at our institution since July of 1993 (types I–III TAA) and when implemented, was supervised in all cases by a dedicated vascular anesthesia team. Details of the clamp and sew technique and the epidural infusion system have been previously reported.[13,14] In brief, the EC system uses an iced saline epidural infusion, which provides for moderate (25-27°C) hypothermia to the spinal cord during the critical period when the aorta is cross-clamped. Patent intercostals vessels in the T9-L1 region were reimplanted by means of a separate inclusion button or were preserved with a beveled anastomosis when technically feasible. Following reperfusion of the lower extremities, EC was discontinued and continuous passive cerebrospinal fluid (CSF) drainage (to keep CSF pressure at 10 mm Hg) was initiated and continued for 48 hours.

Clinical end points

The primary end points of this study included perioperative paraplegia and mortality, long-term freedom from aneurysm-related reintervention, and survival. Perioperative mortality was defined as any death within 30 days of the procedure or any death occurring during the initial hospitalization. All patients were awakened in the operating room for a postoperative baseline neurological examination of the lower extremities. SCI were classified as immediate when noted as the patient awoke from anesthesia, or as delayed when they developed in a patient who was initially neurologically intact. All patients who were expected to have a neurologic deficit postoperatively underwent clinical evaluation by a neurologist. Severity of spinal cord deficit was stratified according to the spinal cord ischemia deficit (SCID) scoring system as previously described.[15] The categories can be summarized as:

SCID I: Flaccid paralysis.
SCID II: Average neurologic muscle grade indicates <50% function.
SCID III: Average neurologic muscle grade indicates >50% function.

Aneurysm-related reintervention included any procedure performed within the initial hospital stay (including re-exploration for bleeding, tracheostomy, additional aortic procedures, etc) and any aortic-related procedures during long-term follow-up. Major pulmonary complications included mechanical ventilation for more than 72 hours postoperatively, reintubation, respiratory failure requiring intensive care unit monitoring, pneumonia, or need for tracheostomy. Significant renal failure included postoperative creatinine levels >3.0 mg/dl in patients with normal baseline levels or need for dialysis. Cardiac complications included myocardial infarction as confirmed by electrocardiogram and appropriate laboratory values or any cardiac event that required intervention. The majority of gastrointestinal complications involved bleeding episodes, and strokes were confirmed using appropriate imaging techniques. Early and long-term freedom from reintervention and survival were determined by a review of office charts and the hospital's computerized medical record. Patients who were last known to be alive but had not been seen in over a year were queried in the Social Security Death Index (SSDI) database to ensure an accurate account of current survival status, while patients who had not been seen for 5 years and had no record in the SSDI were considered lost to follow-up.

Statistical analysis was performed using Chi-square or Fisher's exact test as appropriate for nominal variables. The Student *t* and Wilcoxon tests were used to assess continuous variables. Long-term mortality was determined using Kaplan-Meier survival methods. A Cox proportional hazard model was used to determine multivariate predictors of long-term survival. A P value of $<.05$ was considered significant.

RESULTS

There were 480 patients who underwent open TAA repair during the study interval, of whom 73 (15%) had aneurysms of CD etiology, and 407 (85%) were due to DA. The clinical and demographic features of the cohort stratified by indication are detailed in Table I. Patients with CD were younger, more likely to be male, and have a prior ascending aneurysm repair than patients with DA. The

Table I. Demographic and clinical data for 480 patients undergoing open TA/TAA repair stratified by indication

Variable	*Chronic dissection*	*Degenerative*	P *value*
# patients (%)	73 (15)	407 (85)	
Age, mean ± SD years	64.5 ± 13.5	72.5 ± 8.4	<.001
Male gender (%)	47 (64)	188 (46)	.004
CAD (%)	28 (38)	197 (48)	.11
Smoker (%)	54 (74)	336 (83)	.08
Diabetes (%)	5 (7)	29 (7)	1.00
Hypertension (%)	67 (92)	354 (87)	.25
Family history (%)	17 (23)	26 (6)	<.001
Serum creatinine >1.8 (%)	7 (10)	60 (15)	.24
Prior AAA (%)	15 (21)	91 (22)	.73
Prior ascending aneurysm (%)	17 (23)	16 (4)	<.001
Marfan's syndrome (%)	11 (15)	0	<.001

AAA, Abdominal aortic aneurysm; *CAD,* coronary artery disease; *TA,* thoracic aneurysm; *TAA,* thoracoabdominal aortic aneurysm.

Table II. Aneurysm extent stratified by indication for open repair

Extent	*Chronic dissection*	*Degenerative*	P *value*
# patients (%)	73 (15)	407 (85)	
Descending thoracic aneurysm (%)	14 (19)	38 (9)	<.001
Crawford type I (%)	22 (30)	60 (15)	
Crawford type II (%)	22 (30)	60 (15)	
Crawford type III (%)	13 (18)	150 (37)	
Crawford type IV (%)	2 (3)	99 (24)	

The *P* value is balanced across all extents of aneurysm repair and not specific to one individual type.

distribution of aneurysm extent was different between the two groups ($P < .001$) and is detailed in Table II.

Patients with CD were more likely to be symptomatic upon presentation than those with DA (CD: 47 [65%], DA: 132 [35%], $P < .001$), and elective repair was more common in patients with DA (CD: 43 [59%], DA: 322 [79%], $P = .001$). In addition, patients with CD were more likely to present with rupture than those with DA (CD: 16 [22%], DA: 43 [11%], $P = .001$). The clamp and sew technique was used in 53 (78%) patients with CD versus 355 (93%) patients with DA ($P < .001$), and there was no difference in the use of EC between the two groups when type IV TAA was excluded (CD: 51 [72%], DA: 195 [64%], $P = .20$). The total aortic cross clamp time was similar for both groups (CD: 81 ± 29 minutes, DA: 75 ± 27 minutes, $P = .21$), but the total operative time was longer for the CD group (CD: 366 ± 116 minutes, DA: 306 ± 91 minutes, $P < .001$). Total transfusion requirement (both banked blood and cell saver transfusion) was also significantly higher in the CD group (3.97 L ± 2.2 L, DA: 1.3 L ± .84 L, $P < .001$).

Mortality occurred in the perioperative period in eight (11%) patients with CD and 35 (8.5%) patients with DA ($P = .52$), and there was no difference in SCI (any degree of any deficit) between the two groups (CD: 12 [16%], DA: 45 [11%], $P = .19$). When only elective cases were considered, the perioperative mortality was 7% for both groups ($P = .97$). Immediate SCI occurred in seven (10%) patients with CD and 26 (6%) patients with DA ($P = .32$), and there was no difference in SCID classification which is detailed in Table III. Half (6/12) of the SCI in the CD group occurred in type II TAA, while only 25% (11/45) of the DA were type II TAA. There were two (4%) cases of SCI in type IV TAA in the DA cohort. Intercostal artery reimplantation (as a separate inclusion button) was performed in 54% (65% of patients with Type I and II repairs) of all cases but was more common in patients with CD (CD: 67% vs DA: 52%, $P = .02$). Intercostal artery reimplantation was not protective from SCI in this series, as the SCI rate was 11.7% in patients who had intercostals reimplanted and 12.7% in those who did not ($P = .74$). There was no difference in other complications which are summarized in Table III.

Table III. Details of spinal cord ischemia and other complications stratified by indication for repair

Variable	*Chronic dissection (n = 73)*	*Degenerative (n = 407)*	P *value*
Spinal cord ischemia	12 (16.4%)	45 (11.1%)	.19
Immediate SCI	7/12 (58%)	26/45 (57%)	.97
SCID I (flaccid)	5/12 (42%)	22/45 (49%)	.88
SCID II	4/12 (33%)	12/45 (27%)	
SCID III	3/12 (25%)	11/45 (24%)	
Cardiac complications	10/73 (14%)	64/407 (16%)	.66
Stroke	2/73 (3%)	22/407 (5%)	.34
Pulmonary complications	36/73 (49%)	192/407 (47%)	.74
GI complications	5/73 (7%)	44/407 (11%)	.30
Renal failure	8/73 (11%)	44/407 (11%)	.97

GI, Gastrointestinal; *SCI,* spinal cord ischemia; *SCID,* spinal cord ischemia deficit score.

The average follow-up was 48 months (range, 0-208 months) for the total cohort (CD = 53 months [range, 0-208 months] vs DA = 46 months [range, 0-204 months], $P = .26$). There was no difference in freedom from aneurysm-related reintervention between the two groups; $P = .36$ (Fig 1). There was also no difference in survival, which was 53% and 32% at 5 and 10 years for the CD group and 47% and 17% for DA at the same time points; $P = .07$ (Fig 2). Multivariate predictors of death are summarized in Table IV.

DISCUSSION

This series showed that patients who undergo TAA for CD have a higher preoperative risk profile than those with DA, as they were more likely to present in an emergent/urgent fashion, to have a connective tissue disorder, and to be Crawford types I and II TAA. It has been abundantly documented that extent I and II lesions have a higher risk of SCI, and we and others have documented increased perioperative risks (including increased SCI and mortality)

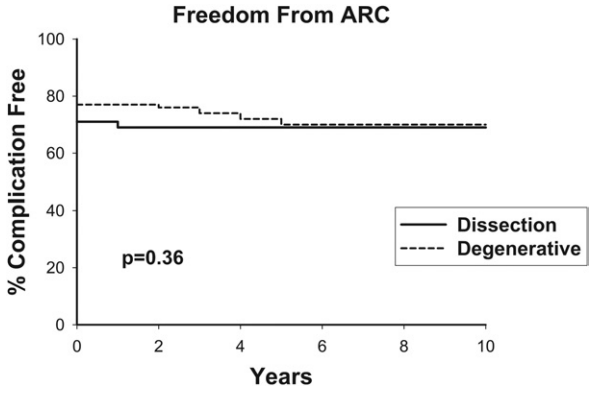

Fig 1. Kaplan-Meier estimate for freedom from aneurysm-related reintervention in patients undergoing TAA stratified by indications (CD vs DA).

Years	*0*	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*
At risk											
CD	73	35	25	18	16	14	13	9	8	5	2
DA	407	189	129	97	62	46	30	25	16	10	7
% without reintervention											
CD	71	69	69	69	69	69	69	69	69	69	69
DA	77	77	76	74	72	70	70	70	70	70	70
SE											
CD	.06	.06	.06	.06	.06	.06	.06	.06	.06	.06	.06
DA	.02	.02	.02	.03	.03	.04	.04	.04	.04	.04	.04

CD, Chronic aortic dissection; *DA,* degenerative aneurysm; *TAA,* thoracoabdominal aortic aneurysm.
P value determined by Mantel-Cox long-rank univariate analysis.

for nonelective operations.[5,9,16-20] However, there was no difference in the 30-day mortality or paraplegia rates between CD and DA in this series. Long-term surveillance showed no difference in freedom from aneurysm-related reintervention, and there was no difference in survival between the two groups with several patients living >15 years. Negative predictors of long-term survival did include increasing age, spinal cord ischemia, preoperative creatinine, and pulmonary function; age was the only variable that differed between the CD and DA cohorts.

The association between CD and perioperative spinal cord ischemia after TAA has evolved over the last 20 years. Crawford's early results with 605 patients (without the use of adjuncts) showed CD to be a significant predictor of spinal cord ischemia on multivariate analysis.[5] However, when Svensson presented the extension of this series to 1509 patients (now with the selective use of adjuncts), chronic dissection was only significant on univariate analysis and was eliminated from the multivariate model.[8] A study from the Cleveland Clinic of patients treated during the same time period as Crawford's original series reported a paraplegia rate of 35% in the 16 patients with dissection. This was not significantly different from the overall 21% incidence of paraplegia in the series but likely would have been if the study was powered adequately.[21] Safi et al reported a contemporary series of TAA repair of which 27% were CD. Their overall neurologic deficit rate was 4.3% with no difference between CD and DA (3.6% vs 4.5%, $P = .58$).[12] A second recent report detailed 660 patients undergoing TAA repair (21% CD) and again there was no difference in SCI between CD and DA (2.9% vs 5.5%).[11] The current study supports these more contemporary series as there was no difference in any degree of SCI between the two groups.

Historically, we have used the clamp and sew technique with EC for spinal cord protection in most TAA repairs, with atrial-femoral bypass being reserved for patients in whom a complex proximal repair is anticipated.[9,22] Indeed, 22% of our CD patients were treated with this adjunct. Other authors have advocated a more liberal approach to the use of atrial-femoral bypass, with contemporary series of TAA for CD reporting the use of distal perfusion in 40% to 80% of patients.[11,12] Jacobs et al reported a spinal cord ischemia rate of 4% in a series of 118 patients with types I and II TAA treated with distal perfusion.[23] We have recently adopted a similar posture of the regular application of atrial-femoral bypass with monitoring of motor-evoked potentials for patients with Crawford type I and II aneurysms regardless of etiology with initial favorable results.

The best way to manage patent intercostal arteries, especially those in the critical region between T8-L1, remains controversial. Historically, the majority opinion (and

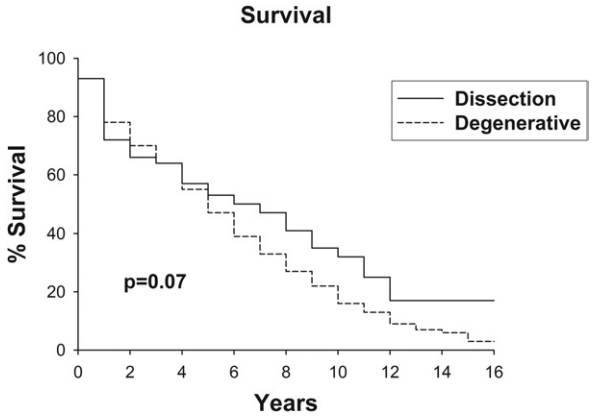

Fig 2. Kaplan-Meier estimate for survival in patients undergoing TAA stratified by indications (CD vs DA).

Years	*0*	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*
At risk											
CD	73	47	40	37	33	28	25	22	18	12	9
DA	407	288	236	212	175	138	107	81	61	46	28
% survival											
CD	93	72	66	64	57	53	50	47	41	35	32
DA	93	78	70	64	55	47	39	33	27	22	16
SE											
CD	.03	.055	.059	.059	.062	.063	.065	.065	.066	.066	.069
DA	.012	.021	.024	.025	.027	.027	.027	.027	.026	.026	.024

P value determined by Mantel-Cox long-rank univariate analysis.
CD, Chronic aortic dissection; *DA*, degenerative aneurysm; *TAA*, thoracoabdominal aortic aneurysm.

Table IV. Multivariate predictors of long-term survival after thoracoabdominal aneurysm repair

Variable	*Coeff*	*95% Confidence interval*	P *value*
Spinal cord ischemia	1.47	1.04-20.9	.03
Age	1.04	1.02-1.06	<.0001
Hypertension	1.71	1.11-2.64	.02
Preop creatinine	1.37	1.2-1.47	<.0001
Chronic obstructive pulmonary disease	1.37	1.0-1.88	.05

consistent with our practice) has been an aggressive posture toward intercostal reconstruction in the T8-L1 segment.[11,12,24,25] Previously, we correlated sacrifice of intercostal vessels therein with an increased risk of SCI.[14] Yet this technical operative adjunct has both a weak evidence base to support its use, and at least in most iterations, is a largely "blind" maneuver (ie, the surgeon genuinely has no data as to the necessity of such intercostal reimplant). A recent large series of TAA repair reported reimplantation of intercostal arteries in 58% of their cohort (80% of type I and II repairs) and found the SCI rate was 5.3% in the group with reimplantation and 13.4% in those that were not reimplanted ($P < .025$).[11] However, Svensson noted, in a prospective study of intercostal preservation, that a majority of separately reimplanted intercostals occlude within the first few hours postoperatively[26] and in the current series (which was mostly clamp and sew), intercostal reimplantation did not decrease SCI. The posture toward intercostal reconstruction has changed over time and a recent review by Acher et al showed that the addition of intercostal artery revascularization to their other protective adjuncts reduced the SCI risk by 75%.[27] The presence of atrial-femoral bypass can complicate intra-operative decision making as it is common to have vigorous back-bleeding from multiple pairs of intercostal arteries, and reimplantation of all vessels would add to the bypass time (and blood turnaround) while providing questionable benefit. Because of this, motor-evoked potentials have been used to predict the need for intercostal reconstruction during atrial-femoral bypass with success.[23,28,29] We now monitor motor-evoked potentials in all patients with types I and II TAA and most type III. After completing the proximal anastomosis, patent intercostal arteries are identified and occluded with Pruit catheters. We then proceed to create the visceral button, checking motor-evoked potentials frequently during this time. If the potentials decrease, dominate intercostal arteries are reimplanted via an inclusion button; if not, they are oversewn.[29] This has resulted in the need for few intercostal reimplantations with minimal paraplegia.

Table V. 30-day mortality results in contemporary series of thoracoabdominal aneurysm repair

Author (Year)	*Patients*	*Mortality (%)*
Svensson (1993)[8]	1509	155 (10)
Coselli (2007)[42]	2286	115 (5)
Safi (2003)[7]	1004	141 (14)
Grabitz et al (1996)[43]	260	37 (14)
Estrera et al (2001)[18]	654	106 (16)
Jacobs (2002)[28]	184	20 (11)
Conrad (2007)[9]	455	39 (8)
Total	6352	613 (9.7)

Despite a variety of advances in anesthesia and the addition of adjunctive techniques, representative large clinical series, including the most recent publications, indicate that the mortality of elective TAA repair remains essentially unchanged from Crawford's initial 8.9%[5] with most series falling in the 7% to 12% range (Table V).[8] Indeed, the results summarized in Table V represent the "best case" scenario, and the "real world" experience is substantially worse. Rigberg et al evaluated 1010 patients from the California Office of Statewide Health Planning and Development database and reported a 30-day elective mortality of 19% that increased to 31% at 1 year.[30] There was also a linear correlation with age at operation so that the 1-year mortality increased from 18% in patients <60 years old to 40% for patients over 80 years of age.[30] Similarly, Cowan et al reviewed 1542 patients who underwent elective TAA repair across a spectrum of hospitals throughout the United States from the National Inpatient Sample. The overall mortality for TAA repair was a sobering 22.3%, with higher volume surgeons having better outcomes than low volume surgeons.[31] Chronic dissection was not individually evaluated in these reports based on large databases, but in multiple single center series, CD was not a predictor of 30-day mortality, nor was there a difference between CD and DA in the current series.[3,11,12]

Little information is available regarding the long-term anatomic follow-up of patients who have undergone TAA repair. Clouse et al from our own institution reported anatomic follow-up in 333 patients with an average surveillance interval of 26 months. They reported a 5-year event-free survival of 71% with chronic dissection not predictive of need for reintervention.[32] In the current report, the 5-year freedom from aneurysm-related reintervention was 69% for CD and 72% for DA, reaffirming that continued surveillance is necessary in this population. We currently obtain a baseline computed tomography scan at the first postoperative visit and then tailor our imaging based on this result. Patients with normal computed tomography scans should be re-evaluated in 4 to 5 years while patients with unaddressed aneurysmal disease should be reimaged sooner.

Few studies have looked at the long-term survival of patients who have undergone TAA repair. Crawford reported a 62% 5-year survival in DA patients, which was significantly higher than the 50% seen in patients with CD ($P < .05$). The current series showed no difference in long-term survival between the two groups despite the fact that the CD patients were an average of 8 years younger than those with DA at the time of TAA repair. In addition, the multivariate negative predictors of long-term survival (age, creatinine, hypertension, and pulmonary disease) in the current series were all predictors of long-term mortality in Crawford's first 605 patients.[5] The presence of increasing numbers of comorbid conditions can naturally be expected to increase overall operative risk. Individual series have demonstrated increased operative risks in patients with antecedent preoperative renal insufficiency.[8,17,21,33-37] These, and the current series reaffirm our posture regarding the importance of chronic renal insufficiency in clinical decision making. Unless there are circumstances where reconstruction of renal artery stenosis (eg, high grade stenosis to a single functioning kidney) has the potential to reverse chronic renal insufficiency, we consider a preoperative creatinine of >1.8 to be a relative contraindication to elective TAA repair. Finally, many of the deaths in our series were identified through the social security death index database and consequently, the cause of death is unknown. As a result, we cannot accurately predict the freedom from aneurysm-related death, which is the ultimate goal of TAA repair regardless of the underlying cause.

CONCLUSIONS

It is clear that advances in operative technique and the application of neuroprotective adjuncts have improved 30-day outcomes after TAA repair for CD such that there is no longer a difference in mortality and paraplegia when compared with DA patients. Evolution of operative strategies, specifically protective adjuncts against SCI (even though these remain in evolution), has likely played the largest role in this transition. However, mortality and paraplegia rates remain significant after TAA, and prevention of aneurysmal degeneration of the false lumen remains the holy grail of the management of Type B aortic dissections. Early reports involving the use of stent grafts in the acute phase show promise[38-41] but further study is needed.

AUTHOR CONTRIBUTIONS

Conception and design: MFC, TB, RC
Analysis and interpretation: MFC, TC, MRC, RC
Data collection: MFC, TC, MRC, VP
Writing the article: MFC, RC
Critical revision of the article: MFC, TB, RC
Final approval of the article: MFC, TC, MRC, VP, TB, RC
Statistical analysis: MFC
Obtained funding: Not applicable
Overall responsibility: MFC

REFERENCES

1. Sueyoshi E, Sakamoto I, Hayashi K, Yamaguchi T, Imada T. Growth rate of aortic diameter in patients with type B aortic dissection during the chronic phase. Circulation 2004;110(11 Suppl 1):II256-61.

2. Juvonen T, Ergin MA, Galla JD, Lansman SL, McCullough JN, Nguyen K, et al. Risk factors for rupture of chronic type B dissections. J Thorac Cardiovasc Surg 1999;117:776-86.
3. Panneton JM, Hollier LH. Nondissecting thoracoabdominal aortic aneurysms. part I. Ann Vasc Surg 1995;9:503-14.
4. Marui A, Mochizuki T, Mitsui N, Koyama T, Kimura F, Horibe M. Toward the best treatment for uncomplicated patients with type B acute aortic dissection: a consideration for sound surgical indication. Circulation 1999;100(19 Suppl):II275-80.
5. Crawford ES, Crawford JL, Safi HJ, Coselli JS, Hess KR, Brooks B, et al. Thoracoabdominal aortic aneurysms: preoperative and intraoperative factors determining immediate and long-term results of operations in 605 patients. J Vasc Surg 1986;3:389-404.
6. Williams GM, Perler BA, Burdick JF, Osterman FA Jr, Mitchell S, Merine D, et al. Angiographic localization of spinal cord blood supply and its relationship to postoperative paraplegia. J Vasc Surg 1991;13: 23-33; discussion:33-5.
7. Safi HJ, Miller CC 3rd, Huynh TT, Estrera AL, Porat EE, Winnerkvist AN, et al. Distal aortic perfusion and cerebrospinal fluid drainage for thoracoabdominal and descending thoracic aortic repair: ten years of organ protection. Ann Surg 2003;238:372-80; discussion:380-1.
8. Svensson LG, Crawford ES, Hess KR, Coselli JS, Safi HJ. Experience with 1509 patients undergoing thoracoabdominal aortic operations. J Vasc Surg 1993;17:357-68; discussion:368-70.
9. Conrad MF, Crawford RS, Davison JK, Cambria RP. Thoracoabdominal aneurysm repair: a 20-year perspective. Ann Thorac Surg 2007;83: S856-861; discussion:S890-2.
10. Jacobs MJ, Meylaerts SA, de Haan P, de Mol BA, Kalkman CJ. Strategies to prevent neurologic deficit based on motor-evoked potentials in type I and II thoracoabdominal aortic aneurysm repair. J Vasc Surg 1999;29:48-57; discussion:57-9.
11. Coselli JS, LeMaire SA, de Figueiredo LP, Kirby RP. Paraplegia after thoracoabdominal aortic aneurysm repair: is dissection a risk factor? Ann Thorac Surg 1997;63:28-35; discussion:35-6.
12. Safi HJ, Miller CC, 3rd, Estrera AL, Huynh TT, Porat EE, Hassoun HT, et al. Chronic aortic dissection not a risk factor for neurologic deficit in thoracoabdominal aortic aneurysm repair. Eur J Vasc Endovasc Surg 2002;23:244-50.
13. Cambria RP, Clouse WD, Davison JK, Dunn PF, Corey M, Dorer D. Thoracoabdominal aneurysm repair: results with 337 operations performed over a 15-year interval. Ann Surg 2002;236:471-9; discussion:479.
14. Cambria RP, Davison JK, Zannetti S, L'Italien G, Brewster DC, Gertler JP, et al. Clinical experience with epidural cooling for spinal cord protection during thoracic and thoracoabdominal aneurysm repair. J Vasc Surg 1997;25:234-41; discussion:241-3.
15. Conrad MF, Ye JY, Chung TK, Davison JK, Cambria RP. Spinal cord complications after thoracic aortic surgery: long-term survival and functional status varies with deficit severity. J Vasc Surg 2008;48:47-53.
16. Cambria R, Davison JK. Spinal cord ischemic complications after thoracoabdominal aortic surgery. In: Gewertz B, Schwartz LB, editors. Surgery of the aorta and its branches. Philadelphia: W B Saunders; 2000.
17. Coselli JS, LeMaire SA, Miller CC 3rd, Schmittling ZC, Koksoy C, Pagan J, et al. Mortality and paraplegia after thoracoabdominal aortic aneurysm repair: a risk factor analysis. Ann Thorac Surg 2000;69:409-14.
18. Estrera AL, Miller CC 3rd, Huynh TT, Porat E, Safi HJ. Neurologic outcome after thoracic and thoracoabdominal aortic aneurysm repair. Ann Thorac Surg 2001;72:1225-30; discussion:1230-1.
19. Safi HJ, Estrera AL, Miller CC, Huynh TT, Porat EE, Azizzadeh A, et al. Evolution of risk for neurologic deficit after descending and thoracoabdominal aortic repair. Ann Thorac Surg 2005;80:2173-9; discussion:2179.
20. Svensson LG, Kouchoukos NT, Miller DC, Bavaria JE, Coselli JS, Curi MA, et al. Expert consensus document on the treatment of descending thoracic aortic disease using endovascular stent-grafts. Ann Thorac Surg 2008;85(1 Suppl):S1-41.
21. Cox GS, O'Hara PJ, Hertzer NR, Piedmonte MR, Krajewski LP, Beven EG. Thoracoabdominal aneurysm repair: a representative experience. J Vasc Surg 1992;15:780-7; discussion:787-8.
22. Cambria RP, Davison JK, Carter C, Brewster DC, Chang Y, Clark KA, et al. Epidural cooling for spinal cord protection during thoracoabdominal aneurysm repair: a five-year experience. J Vasc Surg 2000;31: 1093-102.
23. Jacobs MJ, Mess W, Mochtar B, Nijenhuis RJ, Statius van Eps RG, Schurink GW. The value of motor evoked potentials in reducing paraplegia during thoracoabdominal aneurysm repair. J Vasc Surg 2006;43: 239-46.
24. Shiiya N, Yasuda K, Matsui Y, Sakuma M, Sasaki S. Spinal cord protection during thoracoabdominal aortic aneurysm repair: results of selective reconstruction of the critical segmental arteries guided by evoked spinal cord potential monitoring. J Vasc Surg 1995;21:970-5.
25. Svensson LG, Hess KR, Coselli JS, Safi HJ. Influence of segmental arteries, extent, and atriofemoral bypass on postoperative paraplegia after thoracoabdominal aortic operations. J Vasc Surg 1994;20:255-62.
26. Svensson LG, Patel V, Robinson MF, Ueda T, Roehm JO Jr, Crawford ES. Influence of preservation or perfusion of intraoperatively identified spinal cord blood supply on spinal motor evoked potentials and paraplegia after aortic surgery. J Vasc Surg 1991;13:355-65.
27. Acher CW, Wynn MM, Mell MW, Tefera G, Hoch JR. A quantitative assessment of the impact of intercostal artery reimplantation on paralysis risk in thoracoabdominal aortic aneurysm repair. Ann Surg 2008;248: 529-40.
28. Jacobs MJ, de Mol BA, Elenbaas T, Mess WH, Kalkman CJ, Schurink GW, et al. Spinal cord blood supply in patients with thoracoabdominal aortic aneurysms. J Vasc Surg 2002;35:30-7.
29. Griepp RB, Griepp EB. Spinal cord perfusion and protection during descending thoracic and thoracoabdominal aortic surgery: the collateral network concept. Ann Thorac Surg 2007;83:S865-869; discussion: S890-2.
30. Rigberg DA, McGory ML, Zingmond DS, Maggard MA, Agustin M, Lawrence PF, et al. Thirty-day mortality statistics underestimate the risk of repair of thoracoabdominal aortic aneurysms: a statewide experience. J Vasc Surg 2006;43:217-22; discussion:223.
31. Cowan JA Jr, Dimick JB, Henke PK, Huber TS, Stanley JC, Upchurch GR Jr. Surgical treatment of intact thoracoabdominal aortic aneurysms in the United States: hospital and surgeon volume-related outcomes. J Vasc Surg 2003;37:1169-74.
32. Clouse WD, Marone LK, Davison JK, Dorer DJ, Brewster DC, LaMuraglia GM, et al. Late aortic and graft-related events after thoracoabdominal aneurysm repair. J Vasc Surg 2003;37:254-61.
33. Acher CW, Wynn MM, Hoch JR, Kranner PW. Cardiac function is a risk factor for paralysis in thoracoabdominal aortic replacement. J Vasc Surg 1998;27:821-8; discussion:829-30.
34. Cambria RP, Davison JK, Zannetti S, L'Italien G, Atamian S. Thoracoabdominal aneurysm repair: perspectives over a decade with the clamp-and-sew technique. Ann Surg 1997;226:294-303; discussion:303-5.
35. Conrad M, Crawford RS, Davison JK, Cambria RP. Thoracoabdominal aneurysm repair: a twenty year perspective. An Thorac Sur 2007;83: S856-61; discussion:S890-2.
36. Gilling-Smith GL, Worswick L, Knight PF, Wolfe JH, Mansfield AO. Surgical repair of thoracoabdominal aortic aneurysm: 10 years' experience. Br J Surg 1995;82:624-9.
37. LeMaire SA, Miller CC 3rd, Conklin LD, Schmittling ZC, Coselli JS. Estimating group mortality and paraplegia rates after thoracoabdominal aortic aneurysm repair. Ann Thorac Surg 2003;75:508-13.
38. Dake MD, Kato N, Mitchell RS, Semba CP, Razavi MK, Shimono T, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. N Engl J Med 1999;340:1546-52.
39. Feezor RJ, Martin TD, Hess PJ Jr, Beaver TM, Klodell CT, Lee WA. Early outcomes after endovascular management of acute, complicated type B aortic dissection. J Vasc Surg 2009;49:561-6; discussion:566-7.
40. Greenberg R, Khwaja J, Haulon S, Fulton G. Aortic dissections: new perspectives and treatment paradigms. Eur J Vasc Endovasc Surg 2003;26:579-86.

41. Szeto WY, McGarvey M, Pochettino A, Moser GW, Hoboken A, Cornelius K, et al. Results of a new surgical paradigm: endovascular repair for acute complicated type B aortic dissection. Ann Thorac Surg 2008;86:87-93; discussion:93-4.
42. Coselli JS, Bozinovski J, LeMaire SA. Open surgical repair of 2286 thoracoabdominal aortic aneurysms. Ann Thorac Surg 2007;83:S862-864; discussion:S890-2.
43. Grabitz K, Sandmann W, Stuhmeier K, Mainzer B, Godehardt E, Ohle B, et al. The risk of ischemic spinal cord injury in patients undergoing graft replacement for thoracoabdominal aortic aneurysms. J Vasc Surg 1996;23:230-40.

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DISCUSSION

Dr R. Clement Darling (*Albany, NY*). Mark, those were tremendous results, and obviously you've shown that you've taken care of one of the most severe complications in thoracoabdominal aneurysms in paraplegia.

One question I have is, have you used any other adjuncts besides LA-FA bypass, such as an ax-fem?

And secondly, since you didn't take it out of the variables, such as cooling of the renals, it didn't seem that you showed any benefit, or do you firmly believe that that's an important adjunct to minimizing renal complications in this operation?

Dr Mark F. Conrad. We maintain that renal cooling is an important adjunct because it minimizes the incidence of postoperative renal failure, which can lead to hemodynamic instability and delayed paraplegia. So this adjunct is an important component of the procedure.

We have not used axillofemoral bypasses for distal aortic perfusion. We essentially transitioned from the clamp-and-sew method to the atrial-femoral bypass.

Dr Darling. Do you put the patients on atrial-femoral bypass, or do you have one of the cardiac surgeons do it?

Dr Conrad. Dr Cambria puts the patients on bypass.

Dr Gustavo Oderich (*Rochester, Minn*). With the advent of branched stent grafts, the potential advantages of open repair will be to decrease paraplegia rates by avoiding coverage of the thoracic aorta and reimplanting intercostals, and to provide a durable result with low risk of interventions. I noticed that you used the "Crawford patch technique" to reimplant the visceral arteries. We also used to do that and have a rate of patch aneurysms in 10% to 15% of patients. To achieve the goals mentioned above, we changed our strategy from left heart bypass to hypothermic arrest mostly to provide spinal cord and renal protection. The main reason for is the occurrence of paraplegia for type II extent with heart bypass, and the rates of patch aneurysm. Now we no longer use Crawford patches, and instead prefer complete separate revascularization of each branch. Can you comment on that, please?

Dr Conrad. When you use circulatory arrest on these patients, you are adding a whole new set of complications including additional bleeding and stroke risk, so this has not been our practice. I am not familiar with your personal experience with individual intercostals grafts, but others have reported poor patency with this approach, as many of them have been shown to thrombose on surveillance imaging. So we have continued with the inclusion button/patch technique.

Dr Roy Greenberg (*Cleveland, Ohio*). Mark, I thought that was a really good paper and applaud your results. And I think what you guys have done is what we're all struggling with, which is taking our concurrent treatment groups and comparing them with historical controls, which may be recorded in a prospective database.

You know, our methods of open surgical repair are similar to yours at the Massachusetts General Hospital, which is to use distal perfusion, to use in-line mesenteric perfusion if possible. But the argument that we most frequently get in with vascular surgeons is whether or not these should ever be done without perfusion adjuncts. And I was interested in what your thoughts would be regarding the repair of thoracoabdominal aneurysms, more extensive than type IV. Should such aneurysm repairs ever be considered without perfusion adjuncts?

Dr Conrad. I don't think so. Certainly, with the clamp-and-sew methods, we found that in-line mesenteric shunting made a big difference in the visceral vessel ischemic time and minimizes reperfusion injury. In addition, renal cold has significantly decreased our postoperative renal failure. So I think that for all type I to III aneurysms, some type of distal perfusion adjunct should be used, and I regularly use renal cold with my type IV aneurysms. Thank you.